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Transcriptional activators which differ in their activation potential by more than 3 orders of magnitude are provided. The transactivators are fusions between a DNA binding protein (*e.g.*, a Tet repressor) and minimal transcriptional activation domains derived from Herpes simplex virus protein 16 (VP16). Substitution mutations at amino acid position 442 within the minimal VP16 domain provide transactivators with differing transactivation ability. Moreover, chimeric activation domains comprising both wild type and mutant minimal VP16 domains provide additional variants with differing transactivation ability. Various aspects of the invention pertain to nucleic acid molecules, vectors, host cells, fusion proteins, transgenic and homologous recombinant organisms and methods of regulating gene transcription.